

CARDIOVASCULAR FLASHLIGHT

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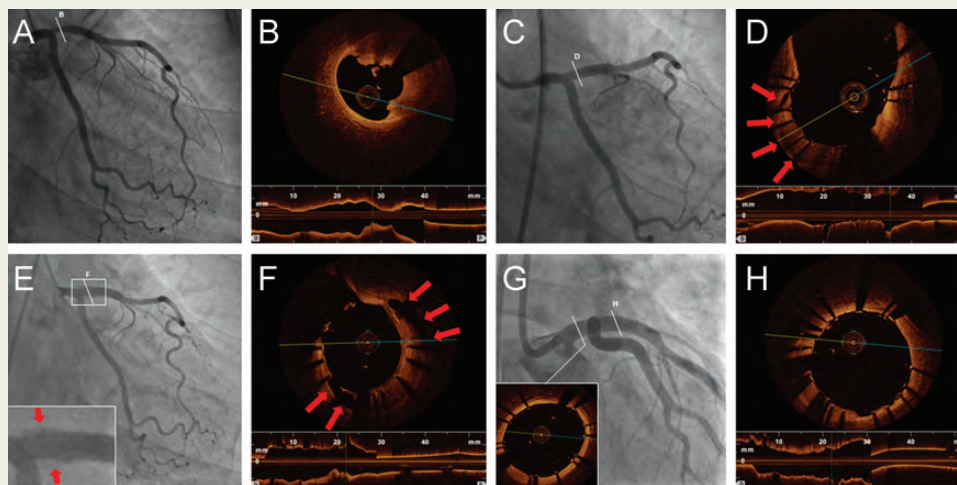
Optical coherence tomography to reveal strut malapposition due to thrombus resolution 3 weeks after acute coronary syndrome

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A 37-year-old male was admitted with a non-ST-elevation myocardial infarction. Coronary angiography and optical coherence tomography (OCT) revealed a 70% stenosis of the ostial left artery descending (LAD) with little thrombotic material but without obvious signs for rupture/dissection (Panels A and B, Supplementary material online, Video S1). After direct stenting with a biolimus-eluting stent (BES,



3.5 × 18 mm, 10 atmospheres), OCT revealed suboptimal strut apposition with compression of an organized thrombus and the underlying plaque (Panels C and D, red arrows). Therefore, two post-dilatations with high-pressure balloon (4.0 × 12 mm, 16 atmospheres) were performed. Afterwards the patient remained asymptomatic and was discharged on dual antiplatelet therapy. Three weeks later the patient was readmitted with new-onset unstable angina. Coronary angiography showed a patent LAD stent but a contrast-filled space underneath the stent struts (Panel E, red arrows). Optical coherence tomography documented homogenous, non-symmetric strut coverage, and acquired malapposition at the proximal stent segment (Panel F, red arrows; Supplementary material online, Video S2). Therefore, direct BES implantation (4.0 × 18 mm, 12 atmosphere) of the LAD and left main with a good final result in OCT after high-pressure balloon post-dilatation (4.0 × 15 mm, 16 atmosphere) was performed (Panels G and H, Supplementary material online, Video S3). The patient's post-procedural recovery was uneventful.

The morphological characteristics of ruptured plaques with subsequent thrombus formation and potential coronary artery spasm in acute coronary syndrome affect strut apposition, which may play a role in stent healing. Here, we present a case in which OCT documented an acquired incomplete stent apposition probably due to resolution of the underlying thrombus. The differential diagnosis would be outward remodelling of the vessel wall as part of a toxic or hypersensitivity reaction to the stent drug or polymer. This seems less likely, as tissue coverage is homogenous, smooth and does not show signs of delayed healing. Our case highlights the potential clinical role of OCT to guide interventional procedures in acute patients.

Supplementary material is available at *European Heart Journal* online.

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